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File 5:Biosis Previews(R) 1969-2003/Oct W4
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4-20-98
4-19-99 p1

Set Items Description

Set	Items	Description
S1	1679	SYNTAXIN
S2	9	SYNTAXIN(2W)INTERACTING
S3	9	SYNIP
S4	8	S3 NOT S2
S5	2260	SNARE
S6	8	SNARE(4W)INTERACTING
S7	8	S6 NOT S2
S8	8	S6 NOT S4
S9	18	AU='MIN JING' OR AU='MIN JINGJUAN'
S10	0	S1 AND S18
S11	2	S1 AND S9
S12	218	E3-E11
S13	20	S12 AND S1
S14	238	E3-E8
S15	2	S14 AND S1
S16	8	S2 NOT S13
S17	11	AU='SYU LI-JYUN'
S18	1	S17 AND S1
S19	0	S15 NOT S13
S20	0	S18 NOT S13

? t s2/7/1-9

2/7/1
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0014504791 BIOSIS NO.: 200300460402
Synip (Syntaxin4 Interacting Protein) and syntaxin4 are expressed in insulin secreting cell and regulate glucose-stimulated insulin secretion.
AUTHOR: Saito Tsugumichi (Reprint); Okada Shuichi; Yamada Eijiro; Ohshima Kihachi; Pessin Jeffrey; Mori Masatomo
AUTHOR ADDRESS: Maebashi, Gunma, Japan**Japan
JOURNAL: Diabetes 52 (Supplement 1): pA374 2003 2003
MEDIUM: print
CONFERENCE/MEETING: 63rd Scientific Sessions of the American Diabetes Association New Orleans, LA, USA June 13-17, 2003; 20030613
SPONSOR: American Diabetes Association
ISSN: 0012-1797 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Poster; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/2
DIALOG(R)File 5:Biosis Previews(R)
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0014165042 BIOSIS NO.: 200300122152
Antisense modulation of %%%syntaxin%%% 4 %%%interacting%%% protein expression
AUTHOR: Freier Susan M (Reprint); Wyatt Jacqueline
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1266 (1): Jan. 7, 2003 2003
MEDIUM: e-file
ISSN: 0098-1133 _(ISSN print)
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Antisense compounds, compositions and methods are provided for modulating the expression of %%%Syntaxin%%% 4 %%%interacting%%% protein. The compositions comprise antisense compounds, particularly antisense

oligonucleotides, targeted to nucleic acids encoding %%%Syntaxin%%% 4 %%%interacting%%% protein. Methods of using these compounds for modulation of %%%Syntaxin%%% 4 %%%interacting%%% protein expression and for treatment of diseases associated with expression of %%%Syntaxin%%% 4 %%%interacting%%% protein are provided.

2/7/3
DIALOG(R)File 5:Biosis Previews(R)
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0013986410 BIOSIS NO.: 200200579921
Identification and characterisation of human %%%syntaxin%%%-4 %%%interacting%%% protein
AUTHOR: Ruddock P E (Reprint); Rutherford A (Reprint); Hardern I (Reprint); Cox C (Reprint); Davies R (Reprint); Marley A (Reprint)
AUTHOR ADDRESS: CVGI Discovery Department, Astrazeneca Pharmaceuticals, Macclesfield, UK**UK
JOURNAL: Diabetologia 45 (Supplement 2): pA 194 August, 2002 2002
MEDIUM: print
CONFERENCE/MEETING: 38th Annual Meeting of the European Association for the Study of Diabetes (EASD) Budapest, Hungary September 01-05, 2002; 20020901
SPONSOR: European Association for the Study of Diabetes
ISSN: 0012-186X
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/4
DIALOG(R)File 5:Biosis Previews(R)
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0013833000 BIOSIS NO.: 200200426511
Ocsyn, a novel %%%syntaxin%%%-%%%interacting%%% protein enriched in the subapical region of inner hair cells
AUTHOR: Safieddine S; Ly C D; Wang Y-X; Wang C Y; Kachar B; Petralia R S; Wenthold R J (Reprint)
AUTHOR ADDRESS: Laboratory of Neurochemistry, National Institute on Deafness and Other Communication Disorders, National Institutes of Health, Bethesda, MD, 20892, USA**USA
JOURNAL: Molecular and Cellular Neuroscience 20 (2): p343-353 June, 2002 2002
MEDIUM: print
ISSN: 1044-7431
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Sensory (hair) cells of the inner ear contain two specialized areas of membrane delivery. The first, located at the cell base, is the afferent synapse where rapid delivery of synaptic vesicles is required to convey information about auditory signals with exceedingly high temporal precision. The second area is at the apex. To accommodate the continuous movement of stereocilia and facilitate their repair, recycling of membrane components is required. Intense vesicular traffic is restricted to a narrow band of cytoplasm around the cuticular plate, which anchors stereocilia. Our previous analyses showed that SNARE proteins (syntaxin 1A/SNAP25/VAMP1) are concentrated at both poles of hair cells, consistent with their involvement in membrane delivery at both locations. To investigate further the molecules involved in membrane delivery at these two sites, we constructed a two-hybrid library of the organ of Corti and probed it with syntaxin 1A. Here we report the cloning of a novel syntaxin-binding protein that is concentrated in a previously uncharacterized organelle at the apex of inner hair cells.

2/7/5
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0013355141 BIOSIS NO.: 200100526980
The %%%syntaxin%%%-%%%interacting%%% synaptic proteins Munc-18 and tomosyn are present in insulin-producing beta-cells and are down-regulated in the diabetic GK rat
AUTHOR: Zhang W (Reprint); Efanov A; Takai Y; Berggren P O; Efendic S; Meister B (Reprint)
AUTHOR ADDRESS: Dept Neuroscience, B3:5, Karolinska Institutet, Stockholm, Sweden**Sweden
JOURNAL: Society for Neuroscience Abstracts 27 (1): p1303 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 31st Annual Meeting of the Society for Neuroscience San Diego, California, USA November 10-15, 2001; 20011110
ISSN: 0190-5295
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The synaptic proteins Munc-18 (also called n-Sec1 or rbSec1) and syntaxin are essential components of the synaptic vesicle fusion complex. Munc-18 interacts with syntaxin and inhibits the formation of core complex. Tomosyn binds to syntaxin and dissociates Munc-18 from syntaxin, thereby to promote membrane fusion. Using immunohistochemistry, we have demonstrated that both Munc-18 and tomosyn, apart from being present in neurons, also exist in beta-cells of rat pancreatic islets and in insulin-secreting HIT-T15 cells. Munc-18 and tomosyn are colocalized in islet cells of rat pancreas. Western blotting revealed a 67 and 130 kDa bands, corresponding to, respectively, Munc-18 and tomosyn in both HIT-T15 cell and RINm5F cell homogenates. Immunoprecipitation showed that both Munc-18 and tomosyn were associated with syntaxin in HIT-T15 cells. Administration of Munc-18 peptide or Munc-18 antiserum to permeabilized HIT-T15 cells resulted in stimulation of insulin secretion. Munc-18 and tomosyn immunoreactivity was markedly weaker in the GK rats as compared to control Wistar rats. In conclusion, our results show that the synaptic protein Munc-18 and tomosyn are present in insulin-secreting beta-cells, that both tomosyn and Munc-18 are associated with syntaxin and that their expression is down-regulated in the diabetic GK rat.

2/7/6
DIALOG(R)File 5:Biosis Previews(R)

0012715634 BIOSIS NO.: 200000433947
P/Q-type calcium channels mediate the activity-dependent feedback of syntaxin-1A
AUTHOR: Sutton Kathy G; McRory John E; Guthrie Heather; Murphy Timothy H; Snutch Terrance P (Reprint)
AUTHOR ADDRESS: Biotechnology Laboratory, Dept Psychiatry, University of British Columbia, Vancouver, BC, V6T 1Z3, Canada**Canada
JOURNAL: Nature (London) 401 (6755): p800-804 Oct. 21, 1999 1999
MEDIUM: print
ISSN: 0028-0836
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Spatial and temporal changes in intracellular calcium concentrations are critical for controlling gene expression in neurons. In many neurons, activity-dependent calcium influx through L-type channels stimulates transcription that depends on the transcription factor CREB by activating a calmodulin-dependent pathway. Here we show that selective influx of calcium through P/Q-type channels is responsible for activating expression of syntaxin-1A, a presynaptic protein that mediates vesicle docking, fusion and neurotransmitter release. The initial P/Q-type calcium signal is amplified by release of calcium from intracellular stores and acts through phosphorylation that is dependent on the calmodulin-dependent kinase, CaM K II/IV, protein kinase A and mitogen-activated protein kinase kinase. Initiation of syntaxin-1A expression is rapid and short-lived, with %%%syntaxin%%%-1A ultimately %%%interacting%%% with the P/Q-type calcium channel to decrease channel

availability. Our results define an activity-dependent feedback pathway that may regulate synaptic efficacy and function in the nervous system.

2/7/7
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0012296320 BIOSIS NO.: 200000014633
The pallid gene encodes a novel, %%%syntaxin%%% 13-%%%interacting%%% protein involved in platelet storage pool deficiency
AUTHOR: Huang Liping; Kuo Yien-Ming; Gitschier Jane (Reprint)
AUTHOR ADDRESS: Howard Hughes Medical Institute, University of California, San Francisco, CA, 94143-0794, USA**USA
JOURNAL: Nature Genetics 23 (3): p329-332 Nov., 1999 1999
MEDIUM: print
ISSN: 1061-4036
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Pallid (pa) is 1 of 13 platelet storage pool deficiency (SPD) mouse mutants. pa animals suffer from prolonged bleeding time, pigment dilution, kidney lysosomal enzyme elevation, serum alpha1-antitrypsin activity deficiency and abnormal otolith formation. As with other mouse mutants of this class, characterization of pa mice suggests a defect in organelle biosynthesis. Here we describe the physical mapping, positional cloning, and mutational and functional analysis of the gene that is defective in pa mice. It encodes a ubiquitously expressed, highly charged 172-amino-acid protein (termed pallidin) with no homology to known proteins. We detected a nonsense mutation at codon 69 of this gene in the pallid mutant. In a yeast two-hybrid screen, we discovered that pallidin interacts with syntaxin 13, a t-SNARE protein that mediates vesicle-docking and fusion. We confirmed this interaction by co-immunoprecipitation assay. Immunofluorescence studies corroborate that the cellular distribution of p allidin overlaps that of syntaxin 13. Whereas the mocha and pearl SPD mutants have defects in Ap-3 (refs 9,10), our findings suggest that pa SPD mutants are defective in a more downstream event of vesicle-trafficking: namely, vesicle-docking and fusion.

2/7/8
DIALOG(R)File 5:Biosis Previews(R)
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0012183481 BIOSIS NO.: 199900443141
%%%Syntaxin%%%-%%%interacting%%% genes regulate volatile anesthetic sensitivity
AUTHOR: Crowder C Michael (Reprint); van Swinderen Bruno (Reprint); Hunt Stephen (Reprint)
AUTHOR ADDRESS: Washington University School of Medicine, Saint Louis, MO, USA**USA
JOURNAL: Anesthesiology (Hagerstown) 91 (3A): pA791 Sept., 1999 1999
MEDIUM: print
CONFERENCE/MEETING: Annual Meeting of the American Society of Anesthesiologists Dallas, Texas, USA October 9-13, 1999; 19991009
SPONSOR: American Society of Anesthesiologists
ISSN: 0003-3022
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/9
DIALOG(R)File 5:Biosis Previews(R)
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0010770752 BIOSIS NO.: 199799404812
Direct interaction of the rat unc-13 homologue Munc13-1 with the N terminus of syntaxin

AUTHOR: Betz Andrea; Okamoto Masaya; Benseler Fritz; Brose Nils (Reprint)
AUTHOR ADDRESS: Max-Planck-Inst. experimentelle Medizin, Abteilung Molekulare Neurobiol., Hermann-Rein-Strasse 3, D-37075 Goettingen, Germany**Germany
JOURNAL: Journal of Biological Chemistry 272 (4): p2520-2526 1997 1997
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: unc-13 mutants in Caenorhabditis elegans are characterized by a severe deficit in neurotransmitter release. Their phenotype is similar to that of the C. elegans unc-18 mutation, which is thought to affect synaptic vesicle docking to the active zone. This suggests a crucial role for the unc-13 gene product in the mediation or regulation of synaptic vesicle exocytosis. Munc13-1 is one of three closely related rat homologues of unc-13. Based on the high degree of similarity between unc-13 and Munc13 proteins, it is thought that their essential function has been conserved from C. elegans to mammals. Munc13-1 is a brain-specific peripheral membrane protein with multiple regulatory domains that may mediate diacylglycerol, phospholipid, and calcium binding. In the present study, we demonstrate by three independent methods that the C terminus of Munc13-1 interacts directly with a putative coiled coil domain in the N-terminal part of syntaxin. Syntaxin is a component of the exocytotic synaptic core complex, a heterotrimeric protein complex with an essential role in transmitter release. Through this interaction, Munc13-1 binds to a subpopulation of the exocytotic core complex containing synaptobrevin, SNAP25 (synaptosomal-associated protein of 25 kDa), and syntaxin, but to no other tested %%%syntaxin%%%-%%%interacting%%% or core complex-interacting protein. The site of interaction in syntaxin is similar to the binding site for the unc-18 homologue Munc18, but different from that of all other known syntaxin interactors. These data indicate that unc-13-related proteins may indeed be involved in the mediation or regulation of synaptic vesicle exocytosis by modulating or regulating core complex formation. The similarity between the unc-13 and unc-18 phenotypes is paralleled by the coincidence of the binding sites for Munc 13-1 and Munc 18 in syntaxin. It is possible that the phenotype of unc-13 and unc-18 mutations is caused by the inability of the respective mutated gene products to bind to syntaxin.

t-snare

binding assay

no competition b. assay

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4/7/1
DIALOG(R)File 5:Biosis Previews(R)

0014504390 BIOSIS NO.: 200300460001
Identification of an Akt2/PKBbeta specific substrate that regulates insulin-stimulated GLUT4 translocation.
AUTHOR: Yamada Eijiro (Reprint); Okada Shuichi; Saito Tsugumichi; Ohshima Kihachi; Pessin Jeffrey; Mori Masatomo
AUTHOR ADDRESS: Maebashi, Gunma, Japan**Japan
JOURNAL: Diabetes 52 (Supplement 1): pA283 2003 2003
MEDIUM: print
CONFERENCE/MEETING: 63rd Scientific Sessions of the American Diabetes Association New Orleans, LA, USA June 13-17, 2003; 20030613
SPONSOR: American Diabetes Association
ISSN: 0012-1797 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

4/7/2
DIALOG(R)File 5:Biosis Previews(R)

0013775110 BIOSIS NO.: 200200368621
Syntaxin 4 is required for the regulated exocytosis of the epithelial

sodium channel (ENaC)
AUTHOR: Banerjee Subhash (Reprint); Olson Diane R (Reprint); Snyder Peter M (Reprint)
AUTHOR ADDRESS: Internal Medicine, University of Iowa College of Medicine, 371 EMRB, Iowa City, IA, 52242, USA**USA
JOURNAL: FASEB Journal 16 (4): pA478 March 20, 2002 2002
MEDIUM: print
CONFERENCE/MEETING: Annual Meeting of the Professional Research Scientists on Experimental Biology New Orleans, Louisiana, USA April 20-24, 2002; 20020420
ISSN: 0892-6638
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Na+ absorption across epithelia is regulated by mechanisms that control the number of Na+ channels (ENaC) at the cell surface. Our aim was to understand mechanisms that regulate ENaC exocytosis. We tested the hypothesis that specific syntaxins are involved in the ENaC regulated exocytosis. We coexpressed ENaC in polarized Fischer rat thyroid cells and measured transepithelial Na+ currents blocked by amiloride (10 muM). We coexpressed ENaC with dominant negative syntaxin mutants lacking the C-terminal membrane anchor, and compared currents to ENaC expressed with green fluorescent protein as a negative control. Mutant syntaxin 4 (syntaxin 4DELTAC) decreased Na+ current by 44% and cAMP-stimulated Na+ current by 47%. Syntaxin 1aDELTAC and syntaxin 3DELTAC did not inhibit baseline or cAMP-stimulated Na+ current. Syntaxin 4 and 3, but not syntaxin 1a, were present in FRT epithelia by western blot. Coexpression of ENaC with %%%synip%%% (a specific syntaxin 4 inhibitory binding protein) nearly abolished baseline and cAMP-stimulated Na+ current. The data suggests that syntaxin 4 mediates the basal and cAMP-regulated exocytosis of ENaC in epithelia.

4/7/3
DIALOG(R)File 5:Biosis Previews(R)

0013305298 BIOSIS NO.: 200100477137
Analysis of insulin signal pathways regulating %%%Synip%%%/syntaxin 4 interactions
AUTHOR: Okada Shuichi (Reprint); Saito Tsugumichi (Reprint); Sato Minoru (Reprint); Ohshima Kimachi (Reprint); Mori Masatomo (Reprint)
AUTHOR ADDRESS: Maebashi, Gunma, Japan**Japan
JOURNAL: Diabetes 50 (Supplement 2): pA405 June, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 61st Scientific Sessions of the American Diabetes Association Philadelphia, Pennsylvania, USA June 22-26, 2001; 20010622
ISSN: 0012-1797
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

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4/7/4
DIALOG(R)File 5:Biosis Previews(R)

0012815641 BIOSIS NO.: 200000533954
Mechanism and regulation of GLUT-4 vesicle fusion in muscle and fat cells
AUTHOR: Foster Leonard J; Klip Amira (Reprint)
AUTHOR ADDRESS: Cell Biology Programme, The Hospital for Sick Children, Toronto, Ontario, M5G 1X8: amira@sickkids.on.ca, Canada**Canada
JOURNAL: American Journal of Physiology 279 (4 Part 1): pC877-C890 October, 2000 2000
MEDIUM: print
ISSN: 0002-9513
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Twenty years ago it was shown that recruitment of glucose transporters from an internal membrane compartment to the plasma membrane led to increased glucose uptake into fat and muscle cells stimulated by insulin. The final step of this process is the fusion of glucose transporter 4 (GLUT-4)-containing vesicles with the plasma membrane. The identification of a neuronal soluble N-ethylmaleimide-sensitive factor attachment protein receptor (SNARE) complex as a requirement for synaptic vesicle-plasma membrane fusion led to the search for homologous complexes outside the nervous system. Indeed, isoforms of the neuronal SNAREs were identified in muscle and fat cells and were shown to be required for GLUT-4 incorporation into the cell membrane. In addition, proteins that bind to nonneuronal SNAREs were cloned and proposed to regulate vesicle fusion. We have summarized the molecular mechanisms leading to membrane fusion in nonneuronal systems, focusing on the role of SNAREs and accessory proteins (Munc18c, %%%synip%%%, Rab4, and VAP-33) in incorporation of GLUT-4 into the plasma membrane. Potential modes of regulation of this process are discussed, including SNARE phosphorylation and interaction with the cytoskeleton.

4/7/5
DIALOG(R)File 5:Biosis Previews(R)

0012307792 BIOSIS NO.: 200000026105
Insulin regulation of GLUT4 vesicle trafficking
AUTHOR: Pessin J E (Reprint)
AUTHOR ADDRESS: University of Iowa, Iowa City, IA, USA**USA
JOURNAL: Growth Hormone and IGF Research 9 (5): p321 Oct., 1999 1999
MEDIUM: print
CONFERENCE/MEETING: 5th International Symposium on Insulin-Like Growth Factors Brighton, England, UK October 31-November 4, 1999. 19991031
SPONSOR: Growth Hormone Research Society
ISSN: 1096-6374
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

4/7/6
DIALOG(R)File 5:Biosis Previews(R)

0012113519 BIOSIS NO.: 199900373179
%%%Synip%%%: A novel insulin-regulated syntaxin 4-binding protein mediating GLUT4 translocation in adipocytes
AUTHOR: Min Jing; Okada Shuichi; Kanzaki Makoto; Elmendorf Jeffrey S; Coker Kenneth J; Ceresa Brian P; Syu Li-Jyun; Noda Yoichi; Saltiel Alan R; Pessin Jeffrey E (Reprint)
AUTHOR ADDRESS: Department of Physiology and Biophysics, University of Iowa, Iowa City, IA, 52242, USA**USA
JOURNAL: Molecular Cell 3 (6): p751-760 June, 1999 1999
MEDIUM: print
ISSN: 1097-2765
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Insulin-stimulated glucose transport and GLUT4 translocation require regulated interactions between the v-SNARE, VAMP2, and the t-SNARE, syntaxin 4. We have isolated a novel syntaxin 4-binding protein, %%%Synip%%%, which specifically interacts with syntaxin 4. Insulin induces a dissociation of the %%%Synip%%%:syntaxin 4 complex due to an apparent decrease in the binding affinity of %%%Synip%%% for syntaxin 4. In contrast, the carboxy-terminal domain of %%%Synip%%% does not dissociate from syntaxin 4 in response to insulin stimulation but inhibits glucose transport and GLUT4 translocation. These data implicate %%%Synip%%% as an insulin-regulated syntaxin 4-binding protein directly involved in the control of glucose transport and GLUT4 vesicle translocation.

4/7/7
DIALOG(R)File 5:Biosis Previews(R)

0012097582 BIOSIS NO.: 199900357242
Characterization of %%%Synip%%%, a novel syntaxin 4 binding protein, and its role in insulin-stimulated GLUT4 vesicle trafficking in 3T3L1 adipocytes
AUTHOR: Elmendorf Jeffrey Scott (Reprint); Okada Shuichi (Reprint); Min Jing (Reprint); Coker Kenneth J (Reprint); Chiang Shian-Huey (Reprint); Khan Ahmir H (Reprint); Saltiel Alan R (Reprint); Pessin Jeffrey E (Reprint)
AUTHOR ADDRESS: Iowa City, IA, USA**USA
JOURNAL: Diabetes 48 (SUPPL. 1): pA78 1999 1999
MEDIUM: print
CONFERENCE/MEETING: 59th Scientific Sessions of the American Diabetes Association San Diego, California, USA June 19-22, 1999; 19990619
SPONSOR: American Diabetes Association
ISSN: 0012-1797
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

4/7/8
DIALOG(R)File 5:Biosis Previews(R)

0012064083 BIOSIS NO.: 199900323743
%%%Synip%%%, a novel Syntaxin4 binding protein that mediates insulin-stimulated GLUT4 translocation
AUTHOR: Min Jing (Reprint); Okada Shuichi (Reprint); Kanzaki Makoto (Reprint); Elmendorf Jeffrey S (Reprint); Coker Kenneth J (Reprint); Ceresa Brian P (Reprint); Syu Li-Jyun (Reprint); Noda Yoichi (Reprint); Saltiel Alan R (Reprint)
AUTHOR ADDRESS: Ann Arbor, MI, USA**USA
JOURNAL: Diabetes 48 (SUPPL. 1): pA11 1999 1999
MEDIUM: print
CONFERENCE/MEETING: 59th Scientific Sessions of the American Diabetes Association San Diego, California, USA June 19-22, 1999; 19990619
SPONSOR: American Diabetes Association
ISSN: 0012-1797
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

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8/7/1
DIALOG(R)File 5:Biosis Previews(R)

0014277154 BIOSIS NO.: 200300233954
Phosphoproteome analysis of capacitated human sperm. Evidence of tyrosine phosphorylation of a kinase-anchoring protein 3 and valosin-containing protein/p97 during capacitation.
AUTHOR: Ficarro Scott; Chertihin Olga; Westbrook V Anne; White Forest; Jayes Friederike; Kalab Petr; Marto Jarrod A; Shabanowitz Jeffrey; Herr John C; Hunt Donald F; Visconti Pablo E (Reprint)
AUTHOR ADDRESS: Dept. of Veterinary and Animal Sciences, University of Massachusetts, 208 Paige Laboratories, Amherst, MA, 01003, USA**USA
AUTHOR E-MAIL ADDRESS: pvisconti@vasci.umass.edu
JOURNAL: Journal of Biological Chemistry 278 (13): p11579-11589 March 28, 2003 2003
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Before fertilization can occur, mammalian sperm must undergo capacitation, a process that requires a cyclic AMP-dependent increase in tyrosine phosphorylation. To identify proteins phosphorylated during capacitation, two-dimensional gel analysis coupled to anti-phosphotyrosine immunoblots and tandem mass spectrometry (MS/MS) was performed. Among the protein targets, valosin-containing protein (VCP), a homolog of the %%%SNARE%%%-%%%interacting%%% protein NSF, and two members of the A kinase-anchoring protein (AKAP) family were found to be tyrosine phosphorylated during capacitation. In addition, immobilized metal affinity chromatography was used to investigate phosphorylation sites in whole protein digests from capacitated human sperm. To increase this chromatographic selectivity for phosphopeptides, acidic residues in peptide digests were converted to their respective methyl esters before affinity chromatography. More than 60 phosphorylated sequences were then mapped by MS/MS, including precise sites of tyrosine and serine phosphorylation of the sperm tail proteins AKAP-3 and AKAP-4. Moreover, differential isotopic labeling was developed to quantify phosphorylation changes occurring during capacitation. The phosphopeptide enrichment and quantification methodology coupled to MS/MS, described here for the first time, can be employed to map and compare phosphorylation sites involved in multiple cellular processes. Although we were unable to determine the exact site of phosphorylation of VCP, we did confirm, using a cross-immunoprecipitation approach, that this protein is tyrosine phosphorylated during capacitation. Immunolocalization of VCP showed fluorescent staining in the neck of noncapacitated sperm. However, after capacitation, staining in the neck decreased, and most of the sperm showed fluorescent staining in the anterior head.

0013459537 BIOSIS NO.: 200200053048
Golgi-to-endoplasmic reticulum (ER) retrograde traffic in yeast requires Dsl1p, a component of the ER target site that interacts with a COPI coat subunit
AUTHOR: Reilly Barbara A; Kraynack Bryan A; VanRheenen Susan M; Waters M Gerard (Reprint)
AUTHOR ADDRESS: Department of Molecular Biology, Princeton University, Princeton, NJ, 08544, USA**USA
JOURNAL: Molecular Biology of the Cell 12 (12): p3783-3796 December, 2001
2001
MEDIUM: print
ISSN: 1059-1524
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: DSL1 was identified through its genetic interaction with SLY1, which encodes a t-%%%SNARE%%%-%%%interacting%%% protein that functions in endoplasmic reticulum (ER)-to-Golgi traffic. Conditional dsl1 mutants exhibit a block in ER-to-Golgi traffic at the restrictive temperature. Here, we show that dsl1 mutants are defective for retrograde Golgi-to-ER traffic, even under conditions where no anterograde transport block is evident. These results suggest that the primary function of Dsl1p may be in retrograde traffic, and that retrograde defects can lead to secondary defects in anterograde traffic. Dsl1p is an ER-localized peripheral membrane protein that can be extracted from the membrane in a multiprotein complex. Immunoisolation of the complex yielded Dsl1p and proteins of apprx80 and apprx55 kDa. The apprx80-kDa protein has been identified as Tip20p, a protein that others have shown to exist in a tight complex with Sec20p, which is apprx50 kDa. Both Sec20p and Tip20p function in retrograde Golgi-to-ER traffic, are ER-localized, and bind to the ER t-SNARE Ufe1p. These findings suggest that an ER-localized complex of Dsl1p, Sec20p, and Tip20p functions in retrograde traffic, perhaps upstream of a Sly1p/Ufe1p complex. Last, we show that Dsl1p interacts with the delta-subunit of the retrograde COPI coat, Ret2p, and discuss possible roles for this interaction.

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0013364113 BIOSIS NO.: 200100535952
Voltage-gated CA2+ channels in cochlear hair cells interact with a novel SH3 domain containing protein
AUTHOR: Hibino H (Reprint); Pironkova R (Reprint); Vologodskaia M (Reprint); Hudspeth A J (Reprint); Lesage F (Reprint)
AUTHOR ADDRESS: Laboratory of Sensory Neuroscience, Howard Hughes Medical Institute, Rockefeller University, New York, NY, USA**USA
JOURNAL: Society for Neuroscience Abstracts 27 (1): p1348 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 31st Annual Meeting of the Society for Neuroscience San Diego, California, USA November 10-15, 2001; 20011110
ISSN: 0190-5295
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Although N- and P-type Ca2+ channels are the predominant site of Ca2+ influx at most fast synapses, L-type Ca2+ channels play a similar role at certain synapses and in secretory cells. In the internal ear, L-type Ca2+ channels that include alpha1D subunits cluster at the presynaptic active zones of hair cells and mediate neurotransmission. Using yeast two-hybrid and GST pull-down assays, we have identified a novel synaptic protein that interacts with the alpha1D subunit. This protein contains SH3 domains that bind to a PXXP motif in the carboxy terminus of the alpha1D subunit. Immunohistochemistry reveals that both proteins are colocalized at the hair cell's presynaptic active zones. The alpha1D-interacting protein additionally associates with the alpha1B subunit of N-type Ca2+ channels in vitro, an interaction that may also occur at synapses in the brain. Because the N-type Ca2+ channel is coupled to the presynaptic protein complex through interactions with %%%SNARE%%% proteins, this alpha1D-%%%interacting%%% protein may provide a linkage between the exocytotic fusion machinery and Ca2+ channels in hair cells as well as neurons.

8/7/4
DIALOG(R)File 5:Biosis Previews(R)
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0012898491 BIOSIS NO.: 200100070330
Regulation of exocytosis by the interaction of the septin CDCrel-1 and tSNARE syntaxin
AUTHOR: Beites C L (Reprint); Salter M W; Trimble W S
AUTHOR ADDRESS: Hosp. Sick Children, Toronto, ON, Canada**Canada
JOURNAL: Society for Neuroscience Abstracts 26 (1-2): pAbstract No.-129.8 2000 2000
MEDIUM: print
CONFERENCE/MEETING: 30th Annual Meeting of the Society of Neuroscience New Orleans, LA, USA November 04-09, 2000; 20001104
SPONSOR: Society for Neuroscience
ISSN: 0190-5295
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: SNARE proteins, localized on vesicles and presynaptic membranes, are thought to mediate the docking and/or fusion of the vesicle with the plasma membrane. However, it is not clearly understood how this process is regulated. In a search for potential SNARE regulators, we have recently identified a novel %%%SNARE%%% %%%interacting%%% protein, the septin CDCrel-1. Septins were first identified as filamentous proteins required for cytokinesis in yeast and more recently in Drosophila. Ten septin isoforms have now been identified in mammals but little is known about their functions. We have shown that CDCrel-1 is predominantly expressed in the brain where it associates with synaptic vesicles and the plasma membrane via its interaction with the SNARE domain of syntaxin 1A.

CDCrel-1, like other septins, contains a GTPase domain. Expression of wildtype CDCrel-1 inhibits secretion upon transfection into insulin-secreting HIT-T15 cells while mutated forms of CDCrel-1 unable to bind GTP have potentiated secretion, suggesting that CDCrel-1 may be regulating vesicle targeting and/or fusion events. We are currently mapping the CDCrel-1 domains important for syntaxin binding. Glycerol gradient centrifugation reveals that the majority of CDCrel-1 fractionates between 4S and 11S. Interestingly, CDCrel-1 can bind syntaxin in a 7S SNARE complex, but this binding is occluded by binding of alphaSNAP. These findings suggest that the CDCrel-1 may regulate the delivery and/or fusion of vesicles to the presynaptic membrane through its interaction with syntaxin. Further studies will be aimed at defining the precise role of CDCrel-1 in targeted secretion.

8/7/5
DIALOG(R)File 5:Biosis Previews(R)

0012651005 BIOSIS NO.: 200000369318
The SNARE Vtila-beta is localized to small synaptic vesicles and participates in a novel SNARE complex
AUTHOR: Antonin Wolfram; Riedel Dietmar; von Mollard Gabriele Fischer (Reprint)
AUTHOR ADDRESS: Zentrum Biochemie und Molekulare Zellbiologie, Abteilung Biochemie II, Universitaet Goettingen, Heinrich-Dueker Weg 12, 37073, Goettingen, Germany**Germany
JOURNAL: Journal of Neuroscience 20 (15): p5724-5732 August 1, 2000 2000
MEDIUM: print
ISSN: 0270-6474
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Specific soluble N-ethylmaleimide-sensitive factor attachment protein (SNAP) receptor (SNARE) proteins are required for different membrane transport steps. The SNARE Vtila has been colocalized with Golgi markers and Vtilb with Golgi and the trans-Golgi network or endosomal markers in fibroblast cell lines. Here we study the distribution of Vtila and Vtilb in brain. Vtilb was found in synaptic vesicles but was not enriched in this organelle. A brain-specific splice variant of Vtila was identified that had an insertion of seven amino acid residues next to the putative %%%SNARE%%%-%%%interacting%%% helix. This Vtila-beta was enriched in small synaptic vesicles and clathrin-coated vesicles isolated from nerve terminals. Vtila-beta also copurified with the synaptic vesicle R-SNARE synaptobrevin during immunoisolation of synaptic vesicles and endosomes. Therefore, both synaptobrevin and Vtila-beta are integral parts of synaptic vesicles throughout their life cycle. Vtila-beta was part of a SNARE complex in nerve terminals, which bound N-ethylmaleimide-sensitive factor and alpha-SNAP. This SNARE complex was different from the exocytic SNARE complex because Vtila-beta was not coimmunoprecipitated with syntaxin 1 or SNAP-25. These data suggest that Vtila-beta does not function in exocytosis but in a separate SNARE complex in a membrane fusion step during recycling or biogenesis of synaptic vesicles.

8/7/6
DIALOG(R)File 5:Biosis Previews(R)

0012080914 BIOSIS NO.: 199900340574
Yeast VSM1 encodes a v-SNARE binding protein that may act as a negative regulator of constitutive exocytosis
AUTHOR: Lustgarten Vardit; Gerst Jeffrey E (Reprint)
AUTHOR ADDRESS: Department of Molecular Genetics, Weizmann Institute of Science, Rehovot, 76100, Israel**Israel
JOURNAL: Molecular and Cellular Biology 19 (6): p4480-4494 June, 1999 1999
MEDIUM: print
ISSN: 0270-7306
DOCUMENT TYPE: Article

RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: We have screened for proteins that interact with v-SNAREs of the late secretory pathway in the yeast Saccharomyces cerevisiae. A novel protein, designated Vsm1, binds tightly to the Snc2 v-SNARE in the two-hybrid system and can be coimmunoprecipitated with Snc1 or Snc2 from solubilized yeast cell extracts. Disruption of the VSM1 gene results in an increase of proteins secreted into the medium but does not affect the processing or secretion of invertase. In contrast, VSM1 overexpression in cells which bear a temperature-sensitive mutation in the Sec9 t-SNARE (sec9-4 cells) results in the accumulation of non-invertase-containing low-density secretory vesicles, inhibits cell growth and the secretion of proteins into the medium, and blocks rescue of the temperature-sensitive phenotype by SNC1 overexpression. Yet, VSM1 overexpression does not affect yeast bearing a sec9-7 allele which, in contrast to sec9-4, encodes a t-SNARE protein capable of forming a stable SNARE complex in vitro at restrictive temperatures. On the basis of these results, we propose that Vsm1 is a novel v-%%%SNARE%%%-%%%interacting%%% protein that appears to act as negative regulator of constitutive exocytosis. Moreover, this regulation appears specific to one of two parallel exocytic paths which are operant in yeast cells.

8/7/7
DIALOG(R)File 5:Biosis Previews(R)

0011572998 BIOSIS NO.: 199800367245
Calcium channels and the %%%SNARE%%% complex %%%interacting%%% for the exocytosis of neurotransmitters
AUTHOR: De Waard Michel (Reprint); Strube Caroline; Villaz Michel
AUTHOR ADDRESS: Inserm U. 464, Faculte Med. Nord, Boulevard Pierre-Dramard, 13916 Marseille Cedex 20, France**France
JOURNAL: M-S (Medecine Sciences) 14 (6-7): p764-770 June-July, 1998 1998
MEDIUM: print
ISSN: 0767-0974
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Citation
LANGUAGE: French

8/7/8
DIALOG(R)File 5:Biosis Previews(R)

0011250342 BIOSIS NO.: 199800044589
Characterization of a novel yeast SNARE protein implicated in Golgi retrograde traffic
AUTHOR: Lupashin Vladimir V; Pokrovskaya Irina D; McNew James A; Waters M Gerard (Reprint)
AUTHOR ADDRESS: Dep. Molecular Biol., Princeton Univ., Princeton, NJ 08544, USA**USA
JOURNAL: Molecular Biology of the Cell 8 (12): p2659-2676 Dec., 1997 1997
MEDIUM: print
ISSN: 1059-1524
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The protein trafficking machinery of eukaryotic cells is employed for protein secretion and for the localization of resident proteins of the exocytic and endocytic pathways. Protein transit between organelles is mediated by transport vesicles that bear integral membrane proteins (v-SNAREs) which selectively interact with similar proteins on the target membrane (t-SNAREs), resulting in a docked vesicle. A novel Saccharomyces cerevisiae SNARE protein, which has been termed Vti1p, was identified by its sequence similarity to known SNAREs. Vti1p is a predominantly Golgi-localized 25-kDa type II integral membrane protein that is essential for yeast viability. Vti1p can bind Sec17p (yeast SNAP) and enter into a Sec18p (NSF)-sensitive complex with the cis-Golgi tSNARE

Sed5p. This Sed5p/Vti1p complex is distinct from the previously described Sed5p/Sec22p anterograde vesicle docking complex. Depletion of Vti1p in vivo causes a defect in the transport of the vacuolar protein carboxypeptidase Y through the Golgi. Temperature-sensitive mutants of Vti1p show a similar carboxypeptidase Y trafficking defect, but the secretion of invertase and gp400/hsp150 is not significantly affected. The temperature-sensitive vti1 growth defect can be rescued by the overexpression of the v-SNARE, Ykt6p, which physically interacts with Vti1p. We propose that Vti1p, along with Ykt6p and perhaps Sft1p, acts as a retrograde v-%%%SNARE%%% capable of %%%interacting%%% with the cis-Golgi t-SNARE Sed5p.

? t s11/7/1-2

11/7/1
DIALOG(R)File 5:Biosis Previews(R)

0012113519 BIOSIS NO.: 199900373179
Synip: A novel insulin-regulated %%%syntaxin%%% 4-binding protein mediating GLUT4 translocation in adipocytes
AUTHOR: %%%Min Jing%%%; Okada Shuichi; Kanzaki Makoto; Elmendorf Jeffrey S; Coker Kenneth J; Ceresa Brian P; Syu Li-Jyun; Noda Yoichi; Saltiel Alan R ; Pessin Jeffrey E (Reprint
AUTHOR ADDRESS: Department of Physiology and Biophysics, University of Iowa, Iowa City, IA, 52242, USA**USA
JOURNAL: Molecular Cell 3 (6): p751-760 June, 1999 1999
MEDIUM: print
ISSN: 1097-2765
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Insulin-stimulated glucose transport and GLUT4 translocation require regulated interactions between the v-SNARE, VAMP2, and the t-SNARE, %%%syntaxin%%% 4. We have isolated a novel %%%syntaxin%%% 4-binding protein, Synip, which specifically interacts with %%%syntaxin%%% 4. Insulin induces a dissociation of the Synip: %%%syntaxin%%% 4 complex due to an apparent decrease in the binding affinity of Synip for %%%syntaxin%%% 4. In contrast, the carboxy-terminal domain of Synip does not dissociate from %%%syntaxin%%% 4 in response to insulin stimulation but inhibits glucose transport and GLUT4 translocation. These data implicate Synip as an insulin-regulated %%%syntaxin%%% 4-binding protein directly involved in the control of glucose transport and GLUT4 vesicle translocation.

11/7/2
DIALOG(R)File 5:Biosis Previews(R)

0012097582 BIOSIS NO.: 199900357242
Characterization of Synip, a novel %%%syntaxin%%% 4 binding protein, and its role in insulin-stimulated GLUT4 vesicle trafficking in 3T3L1 adipocytes
AUTHOR: Elmendorf Jeffrey Scott (Reprint); Okada Shuichi (Reprint); %%%Min%%% %%% Jing%%% (Reprint); Coker Kenneth J (Reprint); Chiang Shian-Huey (Reprint) ; Khan Ahmir H (Reprint); Saltiel Alan R (Reprint); Pessin Jeffrey E (Reprint
AUTHOR ADDRESS: Iowa City, IA, USA**USA
JOURNAL: Diabetes 48 (SUPPL. 1): pA78 1999 1999
MEDIUM: print
CONFERENCE/MEETING: 59th Scientific Sessions of the American Diabetes Association San Diego, California, USA June 19-22, 1999; 19990619
SPONSOR: American Diabetes Association
ISSN: 0012-1797
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

? t s13/7/1-20

13/7/1
DIALOG(R)File 5:Biosis Previews(R)

0014542117 BIOSIS NO.: 200300497145
%%%Syntaxin%%% 4 expression affects glucose transporter 8 translocation and embryo survival.
AUTHOR: Wyman Amanda Hoehn; Chi Maggie; Riley Joan; Carayannopoulos Mary O; Yang Chunmei; Coker Kenneth J; %%%Pessin Jeffrey E%%%; Moley Kelle H (Reprint
AUTHOR ADDRESS: 4911 Barnes-Jewish Hospital Plaza, 6th Floor Maternity, Saint Louis, MO, 63110, USA**USA
AUTHOR E-MAIL ADDRESS: moleyk@msnotes.wustl.edu
JOURNAL: Molecular Endocrinology 17 (10): p2096-2102 October 2003 2003
MEDIUM: print
ISSN: 0888-8809 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Target-soluble N-ethylmaleimide-sensitive factor attachment protein receptors (t-SNAREs) are receptors that facilitate vesicle and target membrane fusion. %%%Syntaxin%%% 4 is the t-SNARE critical for insulin-stimulated glucose transporter 4 (GLUT4)-plasma membrane fusion in adipocytes. GLUT8 is a novel IGF-I/insulin-regulated glucose transporter expressed in the mouse blastocyst. Similar to GLUT4, GLUT8 translocates to the plasma membrane to increase glucose uptake at a stage in development when glucose serves as the main substrate. Any decrease in GLUT8 cell surface expression results in increased apoptosis and pregnancy loss. Previous studies have also shown that disruption of the %%%syntaxin%%% 4 (Stx4a) gene results in early embryonic lethality before embryonic d 7.5. We have now demonstrated that %%%syntaxin%%% 4 protein is localized predominantly to the apical plasma membrane of the murine blastocyst. Stx4a inheritance, as detected by protein expression, occurs with the expected Mendelian frequency up to embryonic d 4.5. In parallel, 22% of the blastocysts from Stx4a+/- matings had no significant insulin-stimulated translocation of GLUT8 whereas 77% displayed either partial or complete translocation to the apical plasma membrane. This difference in GLUT8 translocation directly correlated with one-third of blastocysts from Stx4a+/- mating having reduced rates of insulin-stimulated glucose uptake and 67% with wild-type rates. These data demonstrate that the lack of %%%syntaxin%%% 4 expression results in abnormal movement of GLUT8 in response to insulin, decreased insulin-stimulated glucose uptake, and increased apoptosis. Thus, %%%syntaxin%%% 4 functions as the necessary t-SNARE protein responsible for correct fusion of the GLUT8-containing vesicle with the plasma membrane in the mouse blastocyst.

13/7/2
DIALOG(R)File 5:Biosis Previews(R)

0014504791 BIOSIS NO.: 200300460402
Synip (Syntaxin4 Interacting Protein) and syntaxin4 are expressed in insulin secreting cell and regulate glucose-stimulated insulin secretion.
AUTHOR: Saito Tsugumichi (Reprint); Okada Shuichi; Yamada Eijiro; Ohshima Kihachi; %%%Pessin Jeffrey%%%; Mori Masatomo
AUTHOR ADDRESS: Maebashi, Gunma, Japan**Japan
JOURNAL: Diabetes 52 (Supplement 1): pA374 2003 2003
MEDIUM: print
CONFERENCE/MEETING: 63rd Scientific Sessions of the American Diabetes Association New Orleans, LA, USA June 13-17, 2003; 20030613
SPONSOR: American Diabetes Association
ISSN: 0012-1797 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Poster; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

13/7/3
DIALOG(R)File 5:Biosis Previews(R)

0014504390 BIOSIS NO.: 200300460001
Identification of an Akt2/PKBbeta specific substrate that regulates insulin-stimulated GLUT4 translocation.
AUTHOR: Yamada Eijiro (Reprint); Okada Shuichi; Saito Tsugumichi; Ohshima Kihachi; %%%Pessin Jeffrey%%%; Mori Masatomo
AUTHOR ADDRESS: Maebashi, Gunma, Japan**Japan
JOURNAL: Diabetes 52 (Supplement 1): pA283 2003 2003
MEDIUM: print
CONFERENCE/MEETING: 63rd Scientific Sessions of the American Diabetes Association New Orleans, LA, USA June 13-17, 2003; 20030613
SPONSOR: American Diabetes Association
ISSN: 0012-1797 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

13/7/4
DIALOG(R)File 5:Biosis Previews(R)

0014227049 BIOSIS NO.: 200300185768
Glucose-stimulated insulin secretion is coupled to the interaction of actin with the t-SNARE (target membrane soluble N-ethylmaleimide-sensitive factor attachment protein receptor protein) complex.
AUTHOR: Thurmond Debbie C (Reprint); Gonelle-Gispert Carmen; Furukawa Megumi; Halban Philippe A; %%%Pessin Jeffrey E%%%
AUTHOR ADDRESS: Department of Biochemistry and Molecular Biology, Center for Diabetes Research, Indiana University School of Medicine, Indianapolis, IN, 46202, USA**USA
AUTHOR E-MAIL ADDRESS: dthurmon@iupui.edu
JOURNAL: Molecular Endocrinology 17 (4): p732-742 April 2003 2003
MEDIUM: print
ISSN: 0888-8809 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The actin monomer sequestering agent latrunculin B depolymerized beta-cell cortical actin, which resulted in increased glucose-stimulated insulin secretion in both cultured MIN6 beta-cells and isolated rat islet cells. In perifused islets, latrunculin B treatment increased both first- and second-phase glucose-stimulated insulin secretion without any significant effect on total insulin content. This increase in secretion was independent of calcium regulation because latrunculin B also potentiated calcium-stimulated insulin secretion in permeabilized MIN6 cells. Confocal immunofluorescent microscopy revealed a redistribution of insulin granules to the cell periphery in response to glucose or latrunculin B, which correlated with a reduction in phalloidin staining of cortical actin. Moreover, the t-SNARE (target membrane soluble N-ethylmaleimide-sensitive factor attachment protein (SNAP) receptor) proteins %%%Syntaxin%%% 1 and SNAP-25 coimmunoprecipitated polymerized actin from unstimulated MIN6 cells. Glucose stimulation transiently decreased the amount of actin coimmunoprecipitated with %%%Syntaxin%%% 1 and SNAP-25, and latrunculin B treatment fully ablated the coimmunoprecipitation. In contrast, the actin stabilizing agent jasplakinolide increased the amount of actin coimmunoprecipitated with the t-SNARE complex and prevented its dissociation upon glucose stimulation. These data suggest a mechanism whereby glucose modulates beta-cell cortical actin organization and disrupts the interaction of polymerized actin with the plasma membrane t-SNARE complex at a distal regulatory step in the exocytosis of insulin granules.

13/7/5
DIALOG(R)File 5:Biosis Previews(R)

0013275164 BIOSIS NO.: 200100447003
%%%Syntaxin%%% 4 is required for skeletal muscle insulin-stimulated glucose transport in vivo
AUTHOR: Yang Chunmei (Reprint); Coker Kenneth J (Reprint); Kim Jason (Reprint); Mora Silvia (Reprint); Thurmond Debbie C (Reprint); Shulman Gerald I (Reprint); %%%Pessin Jeffrey E%%% (Reprint
AUTHOR ADDRESS: Iowa City, IA, USA**USA
JOURNAL: Diabetes 50 (Supplement 2): pA524 June, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 61st Scientific Sessions of the American Diabetes Association Philadelphia, Pennsylvania, USA June 22-26, 2001; 20010622
SPONSOR: American Diabetes Association
ISSN: 0012-1797
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

13/7/6
DIALOG(R)File 5:Biosis Previews(R)

0013269622 BIOSIS NO.: 200100441461
Glucose regulates actin dynamics and insulin secretion through the SNARE core complex in pancreatic beta cells
AUTHOR: Thurmond Debbie C (Reprint); %%%Pessin Jeffrey E%%% (Reprint
AUTHOR ADDRESS: Iowa City, IA, USA**USA
JOURNAL: Diabetes 50 (Supplement 2): pA7 June, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 61st Scientific Sessions of the American Diabetes Association Philadelphia, Pennsylvania, USA June 22-26, 2001; 20010622
SPONSOR: American Diabetes Association
ISSN: 0012-1797
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

13/7/7
DIALOG(R)File 5:Biosis Previews(R)

0013199220 BIOSIS NO.: 200100371059
Transmembrane domain length determines intracellular membrane compartment localization of syntaxins 3, 4, and 5
AUTHOR: Watson Robert T; %%%Pessin Jeffrey E%%% (Reprint
AUTHOR ADDRESS: Dept. of Physiology and Biophysics, Univ. of Iowa, Iowa City, IA, 52242, USA**USA
JOURNAL: American Journal of Physiology 281 (1 Part 1): pC215-C223 July, 2001 2001
MEDIUM: print
ISSN: 0002-9513
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Insulin recruits glucose transporter 4 (GLUT-4) vesicles from intracellular stores to the plasma membrane in muscle and adipose tissue by specific interactions between the vesicle membrane-soluble N-ethylmaleimide-sensitive factor attachment protein target receptor (SNARE) protein VAMP-2 and the target membrane SNARE protein %%%syntaxin%%% 4. Although GLUT-4 vesicle trafficking has been intensely studied, few have focused on the mechanism by which the SNAREs themselves localize to specific membrane compartments. We therefore set out to identify the molecular determinants for localizing several %%%syntaxin%%% isoforms, including syntaxins 3, 4, and 5, to their respective intracellular compartments (plasma membrane for syntaxins 3 and 4; cis-Golgi for %%%syntaxin%%% 5). Analysis of a series of deletion and chimeric %%%syntaxin%%% constructs revealed that the 17-amino acid

transmembrane domain of %%%syntaxin%%% 5 was sufficient to direct the cis-Golgi localization of several heterologous reporter constructs. In contrast, the longer 25-amino acid transmembrane domain of %%%syntaxin%%% 3 was sufficient to localize reporter constructs to the plasma membrane. Furthermore, truncation of the %%%syntaxin%%% 3 transmembrane domain to 17 amino acids resulted in a complete conversion to cis-Golgi compartmentalization that was indistinguishable from %%%syntaxin%%% 5. These data support a model wherein short transmembrane domains (ltoreq17 amino acids) direct the cis-Golgi localization of syntaxins, whereas long transmembrane domains (gtoreq23 amino acids) direct plasma membrane localization.

13/7/8
DIALOG(R)File 5:Biosis Previews(R)

0013118831 BIOSIS NO.: 200100290670
%%%Syntaxin%%% 4 heterozygous knockout mice develop muscle insulin resistance
AUTHOR: Yang Chunmei; Coker Kenneth J; Kim Jason K; Mora Silvia; Thurmond Debbie C; Davis Ann C; Yang Baoli; Williamson Roger A; Shulman Gerald I; %%%Pessin Jeffrey E%%% (Reprint
AUTHOR ADDRESS: Department of Physiology and Biophysics, University of Iowa, Iowa City, IA, 52242, USA**USA
JOURNAL: Journal of Clinical Investigation 107 (10): p1311-1318 May, 2001 2001
MEDIUM: print
ISSN: 0021-9738
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: To investigate the physiological function of %%%syntaxin%%% 4 in the regulation of GLUT4 vesicle trafficking, we used homologous recombination to generate %%%syntaxin%%% 4-knockout mice. Homozygotic disruption of the %%%syntaxin%%% 4 gene results in early embryonic lethality, whereas heterozygous knockout mice, Syn4+/-, had normal viability with no significant impairment in growth, development, or reproduction. However, the Syn4+/- mice manifested impaired glucose tolerance with a 50% reduction in whole-body glucose uptake. This defect was attributed to a 50% reduction in skeletal muscle glucose transport determined by 2-deoxyglucose uptake during hyperinsulinemic-euglycemic clamp procedures. In parallel, insulin-stimulated GLUT4 translocation in skeletal muscle was also significantly reduced in these mice. In contrast, Syn4+/- mice displayed normal insulin-stimulated glucose uptake and metabolism in adipose tissue and liver. Together, these data demonstrate that %%%syntaxin%%% 4 plays a critical physiological role in insulin-stimulated glucose uptake in skeletal muscle. Furthermore, reduction in %%%syntaxin%%% 4 protein levels in this tissue can account for the impairment in whole-body insulin-stimulated glucose metabolism in this animal model.

13/7/9
DIALOG(R)File 5:Biosis Previews(R)

0013099806 BIOSIS NO.: 200100271645
Munc18c regulates insulin-stimulated GLUT4 translocation to the transverse tubules in skeletal muscle
AUTHOR: Khan Ahmir H; Thurmond Debbie C; Yang Chunmei; Ceresa Brian P; Sigmund Curt D; %%%Pessin Jeffrey E%%% (Reprint
AUTHOR ADDRESS: Dept. of Physiology and Biophysics, University of Iowa, Iowa City, IA, 52242, USA**USA
JOURNAL: Journal of Biological Chemistry 276 (6): p4063-4069 February 9, 2001 2001
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: To examine the intracellular trafficking and translocation of GLUT4 in skeletal muscle, we have generated transgenic mouse lines that specifically express a GLUT4-EGFP (enhanced green fluorescent protein) fusion protein under the control of the human skeletal muscle actin promoter. These transgenic mice displayed EGFP fluorescence restricted to skeletal muscle and increased glucose tolerance characteristic of enhanced insulin sensitivity. The GLUT4-EGFP protein localized to the same intracellular compartment as the endogenous GLUT4 protein and underwent insulin- and exercise-stimulated translocation to both the sarcolemma and transverse-tubule membranes. Consistent with previous studies in adipocytes, overexpression of the %%%syntaxin%%% 4-binding Munc18c isoform, but not the related Munc18b isoform, in vivo specifically inhibited insulin-stimulated GLUT4-EGFP translocation. Surprisingly, however, Munc18c inhibited GLUT4 translocation to the transverse-tubule membrane without affecting translocation to the sarcolemma membrane. The ability of Munc18c to block GLUT4-EGFP translocation to the transverse-tubule membrane but not the sarcolemma membrane was consistent with substantially reduced levels of %%%syntaxin%%% 4 in the transverse-tubule membrane. Together, these data demonstrate that Munc18c specifically functions in the compartmentalized translocation of GLUT4 to the transverse-tubules in skeletal muscle. In addition, these results underscore the utility of this transgenic model to directly visualize GLUT4 translocation in skeletal muscle.

13/7/10
DIALOG(R)File 5:Biosis Previews(R)

0012680801 BIOSIS NO.: 200000399114
Functional cooperation of two independent targeting domains in %%%syntaxin%%% 6 is required for its efficient localization in the trans-Golgi network of 3T3L1 adipocytes
AUTHOR: Watson Robert T; %%%Pessin Jeffrey E%%% (Reprint
AUTHOR ADDRESS: Department of Physiology and Biophysics, University of Iowa, Iowa City, IA, 52242, USA**USA
JOURNAL: Journal of Biological Chemistry 275 (2): p1261-1268 January 14, 2000 2000
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: To identify the targeting domains of %%%syntaxin%%% 6 responsible for its localization to the trans-Golgi network (TGN), we examined the subcellular distribution of enhanced green fluorescent protein (EGFP) epitope-tagged %%%syntaxin%%% 6/%%%syntaxin%%% 4 chimerae and %%%syntaxin%%% 6 truncation/deletion mutants in 3T3L1 adipocytes. Expression of EGFP-%%%syntaxin%%% 6 resulted in a perinuclear distribution identical to endogenous %%%syntaxin%%% 6 as determined both by confocal fluorescence microscopy and subcellular fractionation. Furthermore, both the endogenous and the expressed EGFP-%%%syntaxin%%% 6 fusion protein were localized to a brefeldin A-insensitive but okadaic acid-sensitive compartment characteristic of the TGN. In contrast, EGFP-%%%syntaxin%%% 6 constructs lacking the H2 domain were excluded from the TGN and were instead primarily localized to the plasma membrane. Although %%%syntaxin%%% 4 was localized to the plasma membrane, %%%syntaxin%%% 6/%%%syntaxin%%% 4 chimerae and %%%syntaxin%%% 6 truncations containing the H2 domain of %%%syntaxin%%% 6 were predominantly directed to the TGN. Importantly, the %%%syntaxin%%% 6 H2 domain fused to the transmembrane domain of %%%syntaxin%%% 4 was also localized to the TGN, demonstrating that the H2 domain was sufficient to confer TGN localization. In addition to the H2 domain, a tyrosine-based plasma membrane internalization signal (YGRL) was identified between the H1 and H2 domains of %%%syntaxin%%% 6. Deletion of this sequence resulted in the accumulation of the EGFP-%%%syntaxin%%% 6 reporter construct at the plasma membrane. Together, these data demonstrate that %%%syntaxin%%% 6 utilizes two distinct domains to drive its specific subcellular localization to the TGN.

13/7/11
DIALOG(R)File 5:Biosis Previews(R)

0012668600 BIOSIS NO.: 200000386913
Discrimination of GLUT4 vesicle trafficking from fusion using a temperature-sensitive Munc18c mutant
AUTHOR: Thurmond Debbie C; %%%Pessin Jeffrey E%%% (Reprint
AUTHOR ADDRESS: Department of Physiology and Biophysics, University of Iowa, Iowa City, IA, 52242-1109, USA**USA
JOURNAL: EMBO (European Molecular Biology Organization) Journal 19 (14): p 3565-3575 July 17, 2000 2000
MEDIUM: print
ISSN: 0261-4189
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: To examine the temporal relationship between pre- and post-docking events, we generated a Munc18c temperature-sensitive mutant (Munc18c/TS) by substitution of arginine 240 with a lysine residue. At the permissive temperature (23degreeC), overexpression of both the wild type (Munc18c/WT) and the R240K mutant inhibited insulin-stimulated GLUT4/IRAP vesicle translocation. However, at the non-permissive temperature (37degreeC) only Munc18c/WT inhibited GLUT4/IRAP translocation whereas Munc18c/TS was without effect. Moreover, Munc18c/WT bound to %%%syntaxin%%% 4 at both 23 and 37degreeC whereas Munc18c/TS bound %%%syntaxin%%% 4 only at 23degreeC. This was due to a temperature-dependent conformational change in Munc18c/TS, as its ability to bind synthaxin 4 and effects on GLUT4 translocation were rapidly reversible while protein expression levels remained unchanged. Furthermore, insulin stimulation of Munc18c/TS-expressing cells at 23degreeC followed by temperature shift to 37degreeC resulted in an increased rate of GLUT4 translocation compared with cells stimulated at 37degreeC. To date, this is the first demonstration that the rate-limiting step for insulin-stimulated GLUT4 translocation is the trafficking of GLUT4 vesicles and not their fusion with the plasma membrane.

13/7/12
DIALOG(R)File 5:Biosis Previews(R)

0012357089 BIOSIS NO.: 200000075402
Munc18c function is required for insulin-stimulated plasma membrane fusion of GLUT4 and insulin-responsive amino peptidase storage vesicles
AUTHOR: Thurmond Debbie C; Kanzaki Makoto; Khan Ahmir H; %%%Pessin Jeffrey%%% %%% E%%% (Reprint
AUTHOR ADDRESS: Department of Physiology and Biophysics, University of Iowa, Iowa City, IA, USA**USA
JOURNAL: Molecular and Cellular Biology 20 (1): p379-388 Jan., 2000 2000
MEDIUM: print
ISSN: 0270-7306
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: To examine the functional role of the interaction between Munc18c and %%%syntaxin%%% 4 in the regulation of GLUT4 translocation in 3T3L1 adipocytes, we assessed the effects of introducing three different peptide fragments (20 to 24 amino acids) of Munc18c from evolutionarily conserved regions of the Sec1 protein family predicted to be solvent exposed. One peptide, termed 18c/pep3, inhibited the binding of full-length Munc18c to %%%syntaxin%%% 4, whereas expression of the other two peptides had no effect. In parallel, microinjection of 18c/pep3 but not a control peptide inhibited the insulin-stimulated translocation of endogenous GLUT4 and insulin-responsive amino peptidase (IRAP) to the plasma membrane. In addition, expression of 18c/pep3 prevented the

insulin-stimulated fusion of endogenous and enhanced green fluorescent protein epitope-tagged GLUT4- and IRAP-containing vesicles into the plasma membrane, as assessed by intact cell immunofluorescence. However, unlike the pattern of inhibition seen with full-length Munc18c expression, cells expressing 18c/pep3 displayed discrete clusters of GLUT4 abd IRAP storage vesicles at the cell surface which were not contiguous with the plasma membrane. Together, these data suggest that the interaction between Munc18c and %%%syntaxin%%% 4 is required for the integration of GLUT4 and IRAP storage vesicles into the plasma membrane but is not necessary for the insulin-stimulated trafficking to and association with the cell surface.

13/7/13
DIALOG(R)File 5:Biosis Previews(R)
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0012307792 BIOSIS NO.: 200000026105
Insulin regulation of GLUT4 vesicle trafficking
AUTHOR: %%%Pessin J E%%% (Reprint
AUTHOR ADDRESS: University of Iowa, Iowa City, IA, USA**USA
JOURNAL: Growth Hormone and IGF Research 9 (5): p321 Oct., 1999 1999
MEDIUM: print
CONFERENCE/MEETING: 5th International Symposium on Insulin-Like Growth Factors Brighton, England, UK October 31-November 4, 1999; 19991031
SPONSOR: Growth Hormone Research Society
ISSN: 1096-6374
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

13/7/14
DIALOG(R)File 5:Biosis Previews(R)
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0012113519 BIOSIS NO.: 199900373179
Synip: A novel insulin-regulated %%%syntaxin%%% 4-binding protein mediating GLUT4 translocation in adipocytes
AUTHOR: Min Jing; Okada Shuichi; Kanzaki Makoto; Elmendorf Jeffrey S; Coker Kenneth J; Ceresa Brian P; Syu Li-Jyun; Noda Yoichi; Saltiel Alan R; %%%Pessin Jeffrey E%%% (Reprint
AUTHOR ADDRESS: Department of Physiology and Biophysics, University of Iowa, Iowa City, IA, 52242, USA**USA
JOURNAL: Molecular Cell 3 (6): p751-760 June, 1999 1999
MEDIUM: print
ISSN: 1097-2765
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Insulin-stimulated glucose transport and GLUT4 translocation require regulated interactions between the v-SNARE, VAMP2, and the t-SNARE, %%%syntaxin%%% 4. We have isolated a novel %%%syntaxin%%% 4-binding protein, Synip, which specifically interacts with %%%syntaxin%%% 4. Insulin induces a dissociation of the Synip: %%%syntaxin%%% 4 complex due to an apparent decrease in the binding affinity of Synip for %%%syntaxin%%% 4. In contrast, the carboxy-terminal domain of Synip does not dissociate from %%%syntaxin%%% 4 in response to insulin stimulation but inhibits glucose transport and GLUT4 translocation. These data implicate Synip as an insulin-regulated %%%syntaxin%%% 4-binding protein directly involved in the control of glucose transport and GLUT4 vesicle translocation.

13/7/15
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0012097582 BIOSIS NO.: 199900357242
Characterization of Synip, a novel %%%syntaxin%%% 4 binding protein, and

its role in insulin-stimulated GLUT4 vesicle trafficking in 3T3L1 adipocytes
AUTHOR: Elmendorf Jeffrey Scott (Reprint); Okada Shuichi (Reprint); Min Jing (Reprint); Coker Kenneth J (Reprint); Chiang Shian-Huey (Reprint); Khan Ahmir H (Reprint); Saltiel Alan R (Reprint); %%%Pessin Jeffrey E%%% (Reprint
AUTHOR ADDRESS: Iowa City, IA, USA**USA
JOURNAL: Diabetes 48 (SUPPL. 1): pA78 1999 1999
MEDIUM: print
CONFERENCE/MEETING: 59th Scientific Sessions of the American Diabetes Association San Diego, California, USA June 19-22, 1999; 19990619
SPONSOR: American Diabetes Association
ISSN: 0012-1797
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

13/7/16
DIALOG(R)File 5:Biosis Previews(R)

0012066066 BIOSIS NO.: 199900325726
Identification of intracellular membrane targeting motifs for syntaxins 4 and 6
AUTHOR: %%%Pessin Jeffery E%%% (Reprint
AUTHOR ADDRESS: Iowa City, IA, USA**USA
JOURNAL: Diabetes 48 (SUPPL. 1): pA7-A8 1999 1999
MEDIUM: print
CONFERENCE/MEETING: 59th Scientific Sessions of the American Diabetes Association San Diego, California, USA June 19-22, 1999; 19990619
SPONSOR: American Diabetes Association
ISSN: 0012-1797
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

13/7/17
DIALOG(R)File 5:Biosis Previews(R)

0011796259 BIOSIS NO.: 199900055919
Regulation of insulin-stimulated GLUT4 translocation by Munc18c in 3T3L1 adipocytes
AUTHOR: Thurmond Debbie C; Ceresa Brian P; Okada Shuichi; Elmendorf Jeffrey S; Coker Kenneth; %%%Pessin Jeffrey E%%% (Reprint
AUTHOR ADDRESS: Dep. Physiol. Biophys., Univ. Iowa, Iowa City, IA 52242, USA**USA
JOURNAL: Journal of Biological Chemistry 273 (50): p33876-33883 Dec. 11, 1998 1998
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Insulin stimulates glucose transporter (GLUT) 4 vesicle translocation from intracellular storage sites to the plasma membrane in 3T3L1 adipocytes through a VAMP2- and %%%syntaxin%%% 4-dependent mechanism. We have observed that Munc18c, a mammalian homolog of the yeast %%%syntaxin%%%-binding protein n-Sec1p, competed for the binding of VAMP2 to %%%syntaxin%%% 4. Consistent with an inhibitory function for Munc18c, expression of Munc18c, but not the related Munc18b isoform, prevented the insulin stimulation of GLUT4 and IRAP/vp165 translocation to the plasma membrane without any significant effect on GLUT1 trafficking. As expected, overexpressed Munc18c was found to co-immunoprecipitate with %%%syntaxin%%% 4 in the basal state. However, these complexes were found to dissociate upon insulin stimulation. Furthermore, endogenous Munc18c was predominantly localized to the plasma membrane and its distribution was not altered by insulin stimulation.

Although expression of enhanced green fluorescent protein-Munc18c primarily resulted in a dispersed cytosolic distribution, co-expression with %%%syntaxin%%% 4 resulted in increased localization to the plasma membrane. Together, these data suggest that Munc18c inhibits the docking/fusion of GLUT4-containing vesicles by blocking the binding of VAMP2 to %%%syntaxin%%% 4. Insulin relieves this inhibition by inducing the dissociation of Munc18c from %%%syntaxin%%% 4 and by sequestering Munc18c to an alternative plasma membrane binding site.

13/7/18
DIALOG(R)File 5:Biosis Previews(R)

0011537548 BIOSIS NO.: 199800331795
Doc2beta: Part of the insulin-stimulated GLUT4 vesicle trafficking machinery in adipocytes
AUTHOR: Thurmond D C; %%%Pessin J E%%%
AUTHOR ADDRESS: Dep. Physiol. and Biophys., Univ. Iowa, Iowa City, IA 52242, USA**USA
JOURNAL: FASEB Journal 12 (8): pA1468 April 24, 1998 1998
MEDIUM: print
CONFERENCE/MEETING: Meeting of the American Society for Biochemistry and Molecular Biology Washington, D.C., USA May 16-20, 1998; 19980516
SPONSOR: American Society for Biochemistry and Molecular Biology
ISSN: 0892-6638
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

13/7/19
DIALOG(R)File 5:Biosis Previews(R)

0011178845 BIOSIS NO.: 199799812905
Osmotic shock stimulates GLUT4 translocation in 3T3L1 adipocytes by a novel tyrosine kinase pathway
AUTHOR: Chen Dong; Elmendorf Jeffrey S; Olson Ann Louise; Li Xiong; Earp H Shelton; %%%Pessin Jeffrey E%%% (Reprint
AUTHOR ADDRESS: Dep. Physiol. Biophysics, Univ. Iowa, Iowa City, IA 52242-1109, USA**USA
JOURNAL: Journal of Biological Chemistry 272 (43): p27401-27410 1997 1997
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Similar to insulin, osmotic shock of 3T3L1 adipocytes stimulated an increase in glucose transport activity and translocation of GLUT4 protein from intracellularly localized vesicles to the plasma membrane. The docking/ fusion of GLUT4 vesicles with the plasma membrane appeared to utilize a similar mechanism, since expression of a dominant interfering mutant of %%%syntaxin%%%-4 prevented both insulin- and osmotic shock-induced GLUT4 translocation. However, although the insulin stimulation of GLUT4 translocation and glucose transport activity was completely inhibited by wortmannin, activation by osmotic shock was wortmannin-insensitive. Furthermore, insulin stimulated the phosphorylation and activation of the Akt kinase, whereas osmotic shock was completely without effect. Surprisingly, treatment of cells with the tyrosine kinase inhibitor, genistein, or microinjection of phosphotyrosine antibody prevented both the insulin- and osmotic shock-stimulated translocation of GLUT4. In addition, osmotic shock induced the tyrosine phosphorylation of several discrete proteins including Cbl, p130-cas, and the recently identified soluble tyrosine kinase, calcium-dependent tyrosine kinase (CADTK). In contrast, insulin had no effect on CADTK but stimulated the tyrosine phosphorylation of Cbl and the tyrosine dephosphorylation of pp125-FAK and p130-cas. These data demonstrate that the osmotic shock stimulation of GLUT4 translocation in adipocytes occurs through a novel tyrosine kinase pathway that is independent of both the phosphatidylinositol 3-kinase and the Akt kinase.

13/7/20
DIALOG(R)File 5:Biosis Previews(R)

0010913320 BIOSIS NO.: 199799547380
%%%Syntaxin%%% 4, VAMP2, and/or VAMP3/cellubrevin are functional target membrane and vesicle SNAP receptors for insulin-stimulated GLUT4 translocation in adipocytes
AUTHOR: Olson Ann Louise; Knight John B; %%%Pessin Jeffrey E%%% (Reprint
AUTHOR ADDRESS: Dep. Physiol. Biophysics, Univ. Iowa, Iowa City, IA 52242-1109, USA**USA
JOURNAL: Molecular and Cellular Biology 17 (5): p2425-2435 1997 1997
ISSN: 0270-7306
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Introduction of the cytoplasmic domain of %%%syntaxin%%% 4, using either recombinant vaccinia virus or single-cell microinjection, resulted in an inhibition of insulin-stimulated GLUT4 but not GLUT1 translocation to the plasma membrane. This was specific for %%%syntaxin%%% 4, since neither the expression of %%%syntaxin%%% 3 nor the expression of a %%%syntaxin%%% 4 mutant in which the vesicle-associated membrane protein (VAMP) binding site was deleted had any significant effect. Consistent with the requirement for a functional VAMP binding site, expression of the cytoplasmic domains of VAMP2 or VAMP3/cellubrevin also resulted in an inhibition of insulin-stimulated GLUT4 translocation. In addition, immunoprecipitation of the expressed %%%syntaxin%%% 4 cytoplasmic domain resulted in an insulin-stimulated increase in the coimmunoprecipitation of GLUT4-containing vesicles. Together, these data demonstrate that %%%syntaxin%%% 4, VAMP2, and/or VAMP3/cellubrevin can function as target membrane and vesicle SNAP receptors, respectively, for insulin-responsive GLUT4 translocation to the plasma membrane.

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